The Privatization of Risk

ABSTRACT

The privatization, or individualization, of risk factor knowledge has been largely responsible for a rising tide of criticism of epidemiology. The current debate seems polarized into 2 sides, those who support and those who attack "risk factor" epidemiology.

This commentary aims to reinvigorate some of Geoffrey Rose's central arguments and show that this debate may miss a key point: a risk factor is a probabilistic concept that applies to an aggregate of individuals, not to a specific individual. Risk factor knowledge compels those in public health to seek actions that shift population distributions of these factors and, to do so, to understand their social, economic, and political determinants. The author links Rose's qualitative distinction between the causes of cases and the causes of incidence to an examination of the conceptual and quantitative limits of "individual risk" estimation.

The attempt to predict individuals' futures on the basis of risk factor profile is especially prominent now with breast cancer. The author suggests reasons why a policy promoting private decision making about risk, while likely ineffective from a population standpoint, is viewed as the only feasible primary prevention option against this disease. (*Am J Public Health.* 2001;91:365–368)

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The privatization, or individualization, of risk factor knowledge has been largely responsible for a rising tide of criticism of epidemiology and preventive medicine.[1–10] This criticism is based on the perception that epidemiologists and other public health researchers, while churning out a seemingly endless torrent of relative risks associated with numerous risk factors, have failed to provide prescriptions for effective public health strategies. The current debate about the practice of epidemiology often seems polarized into 2 sides, those who support and those who attack "risk factor" epidemiology. Some critics of risk factor epidemiology have rather simplistically disallowed the real gains in knowledge about the health effects of behaviors and other lifestyle factors that have come from years of careful research.

An aim of this commentary is to reinvigorate some of Geoffrey Rose's central arguments[11] and show that this current debate may miss a key point: a risk factor is a probabilistic concept that applies to an aggregate of individuals, not to a specific individual. Risk factor knowledge compels those in public health to seek actions that shift population distributions of these factors. I will also discuss how breast cancer may be unique in terms of "public" health concerns: for this disease, a policy promoting private decision making about risk, while likely ineffective from a population standpoint, may be the only feasible primary prevention option.

The Move Toward Individual Risk

The phenomenon of risk privatization, so pervasive in modern epidemiology, reflects the value system now underlying much of public health and preventive medicine in the United States. This system gives primacy to personal autonomy and action and seeks to induce personal behavior change rather than to promote social interventions that often must confront powerful opposing interests.[12] Through numerous and varied channels, including physician counseling, the near-ubiquitous health reports in the lay media, scientific sources, and myriad health-related Web sites, individuals are informed of their individual risk of major diseases on the basis of their personal risk factor profile. These individual risk estimates may pertain to relatively short time periods (5 or 10 years) or to a "lifetime," and they are usually obtained from simple epidemiologic models.

The privatization of risk has important public health implications. Traditionally, risk quantification in public health has taken its shape in large numbers, in statistics regarding average incidence, number needed to treat or screen, and average life expectancy.[1] There are few rules for translating the intrinsically aggregate-level continuous concept of risk into language useful for individuals concerned with dichotomous outcomes and dichotomous decisions about prevention strategies (e.g., use of chemoprevention). Because risk privatization makes risk, and the ability to alter risk, an intrinsic property of the individual,[13] it raises important questions regarding assignation of disease responsibility. There are also important consequences of this individualization for disease prevention. Risk individualization denies the prevention paradox and implies that most epidemiologic risk models are accurate in predicting the future of a specific individual.

Risk Factors Are Poor Screening Tools at the Individual Level

With the shift from infectious disease to chronic disease research that occurred around the middle of the 20th century, the risk factor

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paradigm gained ascendancy among epidemiologists. (The term *risk factor* was coined by Framingham researchers in 1961.[14]) Risk factor logic introduced the notion of probability as a fundamental component of disease causation theories. Risk factors represented the numerous specific causes of disease, but the notion of "cause" was now different from that of the germ theory model that had underlain decades of study of infectious disease. Many of the risk factors hypothesized to be causally related to heart disease and various cancers have only modest associations at the population level. They are neither necessarily nor sufficiently causal at the individual level, by definition as well as by empirical observation. Consequently, the vast majority of chronic disease risk factors, being both unnecessary and insufficient to cause disease, have proved to be quite poor at discriminating at the individual level between those who eventually develop disease (over a certain time period) and those who do not.

This poor discriminatory accuracy of risk factors can be demonstrated statistically. For most diseases studied by epidemiologists, the large majority of individuals will remain disease free over the considered time period, and thus the average estimated "individual" disease risk will be low, often around 0. The relatively few individuals who will develop disease will not receive unusually high estimates of individual risk. This is a quantitative illustration of Rose's point that for most diseases, the large majority of cases arise from the mass of the population with risk factor values (here, "individual" risk estimates) around the average.[11]

The concordance statistic, an index of predictive discrimination of statistical models based on the rank correlation between predicted and observed outcomes,[15] is a widely applicable measure of discriminatory accuracy at the individual level. Potential values of the concordance statistic range from 0.5 to 1.0. The value of the statistic represents the probability that, for a randomly selected diseased individual and a randomly selected nondiseased individual, the diseased individual has the higher estimated disease probability. A concordance statistic of 0.5 for a risk model means that the model performs no better than chance at ranking diseased and nondiseased individuals in terms of estimated probabilities; 50% of the time the diseased person will have the higher estimated probability, while 50% of the time the nondiseased person will. A concordance statistic of 1.0 means that the model performs perfectly at ranking diseased and nondiseased individuals.

Many epidemiologic risk equations produce concordance statistics that are far below 1.0; often they are between 0.50 and 0.70. This is especially true when the risk factors used in the model are associated with only modest relative risks, as is often the case in breast cancer risk models. A risk factor (or set of factors) must be very strongly associated with disease if it is to serve as a worthwhile screening tool, that is, if it is to discriminate well between diseased and nondiseased individuals as reflected by measures of sensitivity and specificity. A given risk factor (or set of factors, where the unexposed group consists of those unexposed to all factors) must have a large relative risk (>20.0) to serve as a useful screening tool.[16]

The Causes of Cases and the Causes of Incidence

In his influential writings about chronic disease risk factors, particularly those for coronary heart disease, Geoffrey Rose developed the theme of the distinction between the causes of disease incidence and the causes of individual cases.[14,17–19] Why did lung cancer incidence rise dramatically in the United States and Britain beginning in the 1930s? Why does breast cancer incidence vary positively with socioeconomic status, and why are incidence rates rising dramatically—to levels found in industrialized societies—in developing countries around the world? Why did coronary heart disease mortality decline in the United States beginning in the early 1960s, and why did the rate of this decline differ by socioeconomic status? These questions about the causes of incidence can be answered by turning to knowledge of the risk factors for the specific diseases and examining the changing distributions of these factors over time and in different population subgroups. However, knowledge that a factor is strongly associated with disease risk can seldom answer the question "Why did this individual get this disease now?" This incapability is both a mathematical reality—following from the modest, probabilistic nature of risk factor–disease associations—and a philosophical argument.[20,21]

Concern about the causes of individual cases vs the causes of population incidence has traditionally been the distinction separating clinical medicine from public health. Knowledge that a factor is associated with increased risk of disease obviously does not translate into the premise that a case of disease will be prevented if a specific individual eliminates exposure; disease pathogenesis at the individual level is a very complex process, as I will elaborate. The misleading message that an individual will prevent a particular disease by altering a particular behavior or exposure (and its converse, that an individual will develop a particular disease if such behavior is not changed) has unfortunately been widely conveyed. Rather, risk factor findings, by necessity couched in probabilistic language, call for aggregate-level policies: if exposure can be eliminated for (say) 1000 individuals, 5 cases (for example) will be averted over a 10-year time period. "Cause" and "prevention," as they pertain to probabilistic risk factor logic, are concepts that apply to an aggregate of individuals, not to a specific individual. Thus, knowledge of proximate risk factors should propel those in public health away from a focus on the individual and compel them to seek ways of shifting population distributions of these factors, by seeking to understand their social, economic, and political determinants.

The "Individual Risk" Approach to Breast Cancer Prevention

Despite the potential pitfalls of relying on assessment of, communication of, and intervention on individual risk to prevent disease, this approach is now prominent with regard to breast cancer prevention. This is probably because, unlike the situation with most other major chronic diseases, *public* health solutions to the problem of breast cancer prevention may be unavailable.

Breast cancer has a unique status among major chronic diseases as a disease of privilege, of women's liberation from their traditional childbearing role.[22] Breast cancer incidence is strongly and positively related to the nutritional status of girls and women.[22,23] Young girls in well-nourished (and now often overnourished) societies experience the onset of menses earlier than young girls who are less well fed, and early menarche is an established risk factor for breast cancer because it signifies higher lifetime exposure to endogenous ovarian hormones. Further, obesity during the postmenopausal period also increases breast cancer risk,[24] through increasing lifetime exposure to estrogens: adipose tissue is the primary site of postmenopausal estrogen production.[25] Breast cancer risk is also strongly and positively related to the degree of women's freedom to choose to reproduce far below traditional and evolutionary levels.[22,23] Nulliparity or low parity, and a late age at first birth, are associated with increased risk of breast cancer, possibly through their detrimental effects on the protective process of breast cell differentiation.[26,27] Attempts to shift social and reproductive norms (e.g., to increase age at menarche or lower age at first birth) to reduce the breast cancer burden would be considered unethical or culturally undesirable in most developed and developing societies; such attempts to alleviate the public health problem of breast cancer could bring with them a net *loss* of public health.

Further, although several "modifiable" lifestyle factors, including postmenopausal hormone use and alcohol consumption, have been linked to modestly increased risk of breast cancer,[28–32] it may be unethical to consider population-based strategies aimed at eliminating or greatly reducing exposure to these factors. On the basis of empiric evidence, both exposures appear to convey a net public health benefit among women: modest alcohol consumption and postmenopausal hormone use are both associated with lower (population) risk of cardiovascular disease,[28,30,33,34] the leading cause of death among older women, and postmenopausal hormones are also associated with reduced risk of osteoporosis[30] and age-related cognitive decline and dementia.[35] Women can also take a chemopreventive agent such as tamoxifen, which has recently been shown to reduce at least short-term breast cancer risk.[36] However, tamoxifen is associated with increased risk of endometrial cancer and deep vein thrombosis/pulmonary embolism, along with more common, though comparatively minor, side effects, and therefore is not recommended for widespread use. Finally, the most extreme choice, prophylactic mastectomy, appears to be highly effective in terms of prevention,[37] but owing to the drastic nature of this option it has so far been reserved for women with strong family histories of the disease or *BRCA1/BRCA2* mutations. I argue that this dilemma, whereby population-based strategies to reduce the burden of one disease, breast cancer, could have net negative effects on overall public health, has helped prompt attempts at preventing cases on an individual-by-individual basis.

Women and their clinicians are increasingly encouraged to use risk estimates derived from statistical risk factor models such as that of Gail et al.[38] (now available to all citizens in the form of the "risk disk," distributed at no cost by the National Cancer Institute and by Zeneca Pharmaceuticals, the maker of tamoxifen) to aid their personal decision making regarding potential prevention options. Women 35 years and older with an estimated 5-year risk of breast cancer of 1.67% according to this risk factor equation are eligible, according to Food and Drug Administration (FDA) guidelines, to consider using tamoxifen prophylactically against breast cancer.

An important question regarding Gail et al.'s model is its accuracy at the probability cutoff of 1.67%.[39] What proportion of women who will develop breast cancer in the near future will have an estimated probability from Gail et al.'s model "high enough" (i.e., above 1.67%) to consider tamoxifen? What proportion of the vast majority of women who will remain free of breast cancer in the near future might nonetheless consider tamoxifen? It is likely that this latter number will be high.[39]

The strategy of preventing breast cancer with tamoxifen, or any other chemopreventive agent, illustrates the prevention paradox[14] well. Many women must engage in the preventive action to prevent disease in only a few. For instance, among 100 women with an estimated 5-year risk of 0.04, approximately 4 women will develop breast cancer over 5 years (assuming the model predictions are well calibrated). If tamoxifen reduces 5-year risk of breast cancer by approximately 50%,[36] 2 of these 4 women will have their breast cancer prevented while 2 women will still develop breast cancer. The remaining 96 women, who will remain free of breast cancer without tamoxifen, will be exposed to increased risk of the adverse outcomes associated with this agent.[40] There is support[40] for the notion that there will be a net *loss* of public health if tamoxifen is used for chemoprevention in all women who are eligible according to FDA guidelines. This reality supports Rose's argument that widespread use of pharmacologic agents for disease prevention is inappropriate.[14] Preventive measures that are implemented in broad segments of the population must be supported by strong evidence indicating their safety in all these segments. Because the "individual" average benefit to each person is small, it can be easily outweighed by the small "individual risk" that accompanies virtually every pharmacologic agent.

Another way of describing the above situation of chemoprevention is in terms of individual risk: each of the 100 women has her "individual" 5-year risk of breast cancer reduced from 0.04 to 0.02. However, this statement is practically meaningless. It disguises the reality that risk refers to a state of *population* health located outside of any one particular individual; it is an aggregate-level concept.

Conclusion

The privatization, or individualization, of the concept of risk raises important practical questions about disease prevention strategies. Most risk factor–disease associations in chronic disease epidemiology are modest (with some notable exceptions, such as smoking and lung cancer and certain occupational exposures and various cancers), and most epidemiologic risk models have correspondingly poor discriminatory accuracy at the individual level. Epidemiologists' ceaseless search for new risk factors, including genes, and the churning out of an endless torrent of relative risks are driven in part by this reality. However, it is likely that the ability to predict the futures of individuals will always remain out of reach, despite ever-increasing knowledge about alleged independent factors or genes that may elevate disease risk in exposed groups.

While the disease of breast cancer may be unique in terms of "public" health concerns, in that no beneficial population-wide prevention strategies are readily apparent, it ironically demonstrates the logic of population-wide approaches. Suggested primary prevention strategies, including chemoprevention or avoidance of postmenopausal hormones, that are directed at supposedly "high-risk" women will not have a large impact on disease burden *unless* the strategy becomes widespread. That is, unless a large proportion of women participate in such strategies, few cases of disease will actually be prevented.

"Public" health today increasingly means education about "personal" risk for a variety of diseases. There is a growing belief in our society that individuals can, and should, exert fundamental control over their future by rationally acting to lower personal risk of a variety of diseases. This focus on individual risk calculation and communication can have positive consequences, such as promotion of healthy lifestyles in segments of the population able to obtain, and voluntarily act on, accurate health risk information. However, there are substantial negative consequences as well. Most important, there is little precedent for relying on communication about individual risk to meaningfully reduce chronic disease burden. Even a cursory reading of the history of chronic disease trends will demonstrate that favorable population-wide changes in risk factors, and resulting decreases in disease incidence, have rarely resulted from individual risk calculations made simultaneously by key "high-risk" individuals in the population.

There are potential dangers in designating the individual the *sole* locus of "risk" and thus the locus of responsibility for "risk reduction." One danger is the amplification of existing socioeconomic health inequities, as individuals in lower socioeconomic strata are less likely to have regular contact with the health care system, to comprehend the arithmetic behind risk information, and to have the psychologic, social, and economic resources needed to voluntarily alter the factors contributing to their "personal" risk.

Another possible negative effect is the assignation of personal responsibility for illness. Ironically, while there has often been a moralistic tendency to blame individuals for their own poor health outcomes,[14] supposedly "progressive" public health research is now being used, inappropriately, to justify such individual accountability. The labeling of these risk factors as the "causes" of individual cases of disease, and the implication that responsible individuals who avoid such risk factors will prevent their own case of disease, represent strong denials of the inability of statistics and medical science to predict the futures of individuals. Further, the equating of risk factors

with the causes of individual cases fosters an indifference to the social determinants of risk factor distributions and thus contributes to ineffectual disease prevention policies at the population level. □

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